CLAIMS:

1. Isolated and purified human 12-lipoxygenase from human tissue, said isolated and purified human 12-lipoxygenase being distinct from that found in human platelets and distinct from human 15-lipoxygenase.

2. The isolated and purified human 12-lipoxygenase of claim 1, wherein said 12-lipoxygenase is from human vascular smooth muscle cells, adrenal cells, endothelial cells or monocytes.

3. A method for diagnosing a disease state in a patient in which the 12-lipoxygenase of claims 1-2 or 12-HETE is an etiological agent which comprises:

(i) obtaining a physiological specimen from said patient; and

(ii) determining whether the etiological agent or antibodies to said etiological agent are present in said specimen.

4. The method of claim 3 in which said disease state is an inflammatory or autoimmune condition atherosclerosis, cancer growth, cancer metastasis, dysfunctional secretion of insulin in non-insulin dependent diabetics, glucose-induced oxidative stress or the development of end-organ dysfunction or damage.

5. The method of claim 4, wherein said disease state is atherosclerotic vascular disease, human breast cancer, or Type II diabetes.

6. A method for treating a patient suffering form a disease state in which hl 12-lipoxygenase or 12-HETE is an etiological agent which comprises administering to said human patient an hl 12-lipoxygenase inhibitor in an amount effective to retard or inhibit expression of said hl 12-lipoxygenase.

7. The method of claim 6, wherein said disease state is Type II diabetes or breast cancer.

8. The method of claim 6, wherein said disease state is non-insulin dependent diabetes mellitus and the human 12-lipoxygenase inhibitor is administered in an amount therapeutically effective to enhance the ability of insulin to induce glucose transport in muscle and the secretion of insulin in pancreatic islets.

9. The method of claim 6, in which said inhibitor is NDGA, CDC, panaxynol, biacalein, pioglitazone, aminoguanidine or a ribozyme which cleaves hl 12-lipoxygenase mRNA.

10. A method for inhibiting or reducing vascular endothelial growth factor production in a human patient which comprises administering to said patient an hl 12-lipoxygenase inhibitor in an amount effective to retard or inhibit the expression of hl 12-lipoxygenase.

11. A method for the treatment of a human patient suffering from a cytokine mediated autoimmune, inflammatory or atherosclerotic disorder which comprises administering to said patient a human 12-lipoxygenase inhibitor in an amount therapeutically effective to mediate the action of said cytokine on human vascular smooth muscle.

12. The method of claim 11 in which said cytokine is interleukin 1, interleukin 4 or interleukin 8.

13. A method for reducing monocyte binding to human endothelial cells which comprises administering to a patient in need thereof an inhibitor of human 12-lipoxygenase in a therapeutically effective amount.

14. The method of claims 10, 11, 12 or 13, wherein said inhibitor is NDGA, CDC, panaxynol, baicalein, pioglitazone, aminoguanidine or a ribozyme which cleaves hl 12-lipoxygenase mRNA.

15. A method for generating lipid mediators which activate signal transduction pathways associated with

inflammatory and autoimmune conditions, atherosclerosis, cancer growth and metastasis, said lipid mediators being hydroperoxides, kinases, mitogen activated kinases, transcription factors, and oncogenes which method comprises administering to a mammal in need thereof a therapeutically effective amount of arachidonic or linoleic acid, wherein said human 12-lipoxygenase utilizes said acid to generate such a lipid mediator.

16. The method of claim 15 in which said lipid mediator is a hydroperoxide, a mitogen activated kinase or NFkB.

17. A method for increasing the activity and expression of human 12-lipoxygenase which comprises administering to a human patient a therapeutically effective amount of an inflammatory cytokine, a growth factor or angiotensin II.

18. The method of claim 17 in which said inflammatory cytokine is interleukin-1.

19. The method of claim 17 in which said growth factor is platelet derived growth factor.

20. A method for increasing the activity and expression of 12-lipoxygenase mRNA and protein in human aortic smooth muscle cells which comprises treatment of said cells with angiotensin II in an amount effective to increase said activity and expression.

21. A method for the reduction of angiotensin II related cardiovascular disease which comprises administration to a patient in need thereof a therapeutically effective amount of a drug effective to blockade the human 12-LO pathway.

22. A method for inhibiting the proliferation of breast cancer tissue in a human patient which comprises administering to said patient a therapeutically effective

amount of a drug which inhibits human 12-LO expression or activation.

23. The method of claim 22 in which said human 12-lipoxygenase inhibitor is NDGA, CDC, panaxynol, baicalein, pioglitazone, aminoguanidine or a ribozyme which cleaves hl 12-lipoxygenase mRNA.

24. The method of claim 22 in which the breast cancer cell growth and development is basal, epidermal growth factor-induced or estrogen induced.

25. A method for mediating breast cancer cell growth and development which comprises administering to a patient in need thereof a therapeutically effective amount of a human 12-lipoxygenase pathway inhibitor.

26. The method of claim 25 in which said human 12-lipoxygenase inhibitor is NDGA, CDC, panaxynol, baicalein, pioglitazone, aminoguanidine or a ribozyme which cleaves hl 12-lipoxygenase mRNA.

27. The method of claim 25 in which the breast cancer cell growth and development is basal, epidermal growth factor-induced or estrogen induced.

28. A method for monitoring cytokine induced vascular smooth muscle cell migration and proliferation observed in an atherosclerosis which comprises measuring the change in human 12-lipoxygenase mRNA or protein expression by said cells upon treatment with a cytokine wherein an increase in said expression is indicative of said cell migration and proliferation.

29. A method for treating a patient having a disease state in which 12-HETE is an etiological agent which comprises decreasing mitogenic activity in said patient.

30. The method of claim 29, wherein the decrease in mitogenic activity results from reducing ERK, JAK and/or JNK activity in said patient.

31. The method of claim 30, wherein JNK activity is reduced by decreasing PAK activity.

32. The method of claim 29, wherein the mitogenic activity is decreased by administering a therapeutically effective amount of a 12-LO inhibitor to the patient sufficient to decrease mitogenic activity.

33. The method of claim 29, wherein the disease state is Type II diabetes or breast cancer.

34. A method for treating a patient having a disease state in which VEGF is an etiological agent which comprises decreasing the amount of VEGF in said patient.

35. The method of claim 34, wherein the disease state is proliferative diabetic retinopathy or accelerated vascular disease associated with diabetes.

36. The method of claim 34, wherein the amount of VEGF is decreased by administering to the patient a therapeutically effective amount of a 12-LO inhibitor sufficient to decrease VEGF in the patient.

37. A method for increasing insulin receptor phosphorylation in patients having Type II diabetes which comprises administering to the patient a therapeutically effective amount of a 12-LO inhibitor sufficient to inhibit 12-LO pathway products from inhibiting insulin receptor phosphorylation.

38. A method for reducing 12-LO gene expression and 12-HETE levels in a patient which comprises administering to the patient a therapeutically effective amount of Mg.